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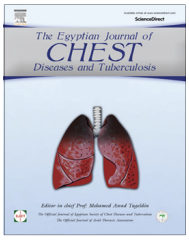

Tuberculous pleural effusion – relapse or re-infection? Follow up of a case report and review of the literature

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KEYWORDS

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Abstract *Introduction:* This is a case report of pleural effusion with acute drug reactions to anti-tubercular drugs.

Case presentation: A 45 year old female patient had the typical signs and symptoms of tuberculosis with continuous coughing, breathlessness and protruded abdomen. The USG abdomen showed fluid in the left pleural space. Thoracocentesis was carried out thrice at an interval of 15 days and about 5.15 L were aspirated. The symptoms of dyspnea and cough were relieved. There was acute drug reaction after starting the ATT. The blood pressure was very high requiring hospital admission. The patient was monitored during the entire course of treatment. No fluid in the bilateral pleural spaces was observed in USG after 6 months of treatment.

This patient had spinal tuberculosis 8 years ago and had recovered following ATT. Therefore, it is difficult to say whether pleural effusion was due to relapse of a previous infection or a re-infection. It is also not known whether TB patients remain susceptible to yet another infection in some other extra-pulmonary site.

Conclusion: Abdominal TB should be suspected in patients with fever, abdominal pain and ascites. Sputum induction (in addition to pleural fluid) for acid-fast bacilli and culture is a recommended procedure in all patients with TB pleurisy.

This condition carries good prognosis, if promptly diagnosed and treated. A reasonable management strategy for pleural TB would be to initiate a four-drug regimen and perform a therapeutic thoracocentesis in patients with large, symptomatic effusions. Prolonged follow-up is essential in cases of pleural effusion, as in the presented case.

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Introduction

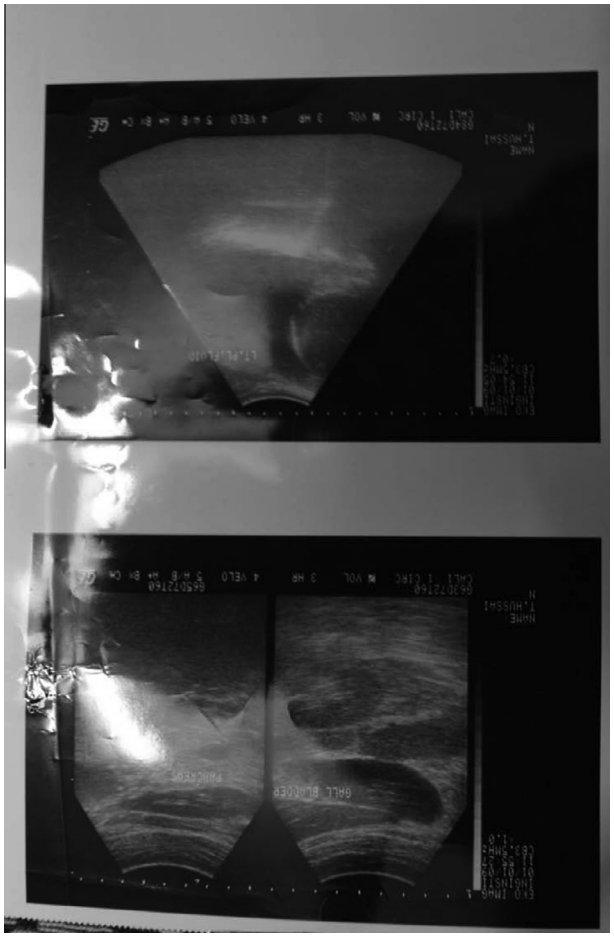
Consent

A written informed consent was obtained from the patient for publication of this case report and accompanying images.

Case presentation

This is a case of pleural effusion due to tuberculosis. A 45 year old female patient had the following signs and symptoms. She was having low grade night fever, weight loss of 3–4 kgs, and cough that lasted. The fever and cough continued even after standard antibiotics and paracetamol for 2 weeks. This was followed by breathlessness, protruded abdomen, discomfort while sleeping, etc. This condition resulted in feeling ashamed while going out. Every day, the abdomen looked even more bigger until one day a reddish outline was observed. The abdomen appeared tight to touch, stretched and shiny. The patient coughed continuously and breathing sounds could be heard while lying down. The doctor advised ultrasonography (USG) along with other tests. USG abdomen showed fluid in left pleural space (Figs. 1 and 2). Thoracocentesis was advised and was carried out thrice at an interval of 15 days and about 5.15 L [2.5 L, 2.2 L and 450 ml fluid at each sitting] were aspirated. The symptoms of dyspnea and cough were relieved. The fluid was sent for Gram staining and culture and TB markers (adenosine deaminase >45 IU/L). Cytology revealed plenty of lymphocytes and occasional macrophages but mesothelial/malignant cells were not found. FNAC of supra-clavicular nodule showed blood only. ADA of pleural fluid was 84 U/L [normal <30 U/L, suspect 30–4 U/L, strong suspect >40–<60, positive >60 U/L]. Uric acid – 8.69, cholesterol – 222, Urea – 39 mg/dl, creatinine – 1.4, bilirubin – 0.2, total bilirubin – 0.9, SGOT (AST) – 48 [5–40 IU/L], SGPT (ALT) – 43 [5–50 IU/L], serum alkaline phosphatase – 432 IU/L [100–300 IU/L]. The smear of pleural fluid showed few pus cells but no bacteria. The protein content was 5.8 g/dl and LDH was 368 U/L [250–450]. Anti-TB treatment was started. A few weeks after starting the ATT, there was severe drug reaction followed by vomiting. The blood pressure was very high. The patient was admitted in the TB ward and anti-hypertensives as well as diuretics were administered. The patient became stable after 12 h and all blood tests were normal. A fresh batch of ATT drugs along with multi-vitamins were administered and the patient was discharged from the ward after a week. The treatment continued for 6 months and the patient was advised X-ray, DC, TLC, LFT, Hb and USG after completion of treatment. USG abdomen revealed no fluid in the bilateral pleural spaces and all other parameters of blood tests were within normal range.

This patient had spinal tuberculosis about 8 years ago, wherein lytic areas of bone destruction in D9, D10, D11 and L2, L3, L5 vertebral bodies were observed in CT scan images. Hypodense collection was seen in the pre- and para- vertebral spaces at D9–D11 levels. She was treated with standard anti-tuberculosis treatment, advised bed rest, restricted movement and good nutritional support along with lumbo-sacral braces while ambulant and recovered from grade III paraplegia [16].



Figures 1 and 2 The left pleural effusion of the patient.

Discussion

Pleural effusion is defined as an abnormal accumulation of fluid in the pleural space. It is not a disease but rather a complication of an underlying illness [1,2]. Excess fluid results from the disruption of the equilibrium that exists across pleural membranes. Pleural effusion is an indicator of a pathologic process that may be of primary pulmonary origin or of an origin related to another organ system or to systemic disease [3,4]. It may occur in the setting of acute or chronic disease and is not a diagnosis in itself [5,6].

It is a common clinical finding with a wide range of causes, but develops most frequently as a part of the decompensation of previously asymptomatic chronic liver disease. The clinical manifestations of ascites vary from an asymptomatic patient to patients complaining of increased abdominal girth, early satiety, and respiratory distress depending on the amount of fluid accumulated in the abdomen [7]. On physical examination the presence of ascites is suggested by the following findings: abdominal distension, bulging flanks, tympany of the top, fluid wave, shifting dullness, puddle sign.

Ultrasonography is performed to detect or exclude the presence of fluid, if the physical examination is not definitive, since abdominal USG can detect small amounts of fluid as 100 ml [8]. In thoracocentesis procedure, a needle is inserted through

the back of the chest wall in the sixth, seventh, or eighth intercostal space on the mid-axillary line, into the pleural space and fluid is drained. The fluid is then evaluated for the chemical composition including protein, lactate dehydrogenase (LDH), albumin, amylase, pH, glucose, Gram stain and culture to identify possible bacterial infections, cell count and differential, cytopathology to identify cancer cells, some infective organisms and other tests as suggested by the clinical situation namely lipids, fungal culture, viral culture and specific immunoglobulins [9–13].

Tuberculous pleural effusion is one of the most common forms of extra-pulmonary tuberculosis (TB). The immediate cause of the effusion is a delayed hypersensitivity response to mycobacterial antigens in the pleural space. Abdominal paracentesis with analysis of the ascitic fluid should be done in patients with new onset ascites, those requiring hospitalization because of ascites and those whose condition deteriorates during hospitalization [14]. In areas with high TB prevalence, pleural fluid adenosine deaminase (ADA) levels greater than 40 U/l argue strongly for TB; in contrast, low levels of pleural ADA have high negative predictive value in low-prevalence countries [15]. The specificity of this enzyme increases if only lymphocytic exudates are considered. The shortcoming of the ADA test is its inability to provide culture and drug sensitivity information, which is paramount in countries with a high degree of resistance to anti-TB drugs. This patient had spinal tuberculosis about 8 years ago and had recovered following ATT [16]. Therefore, it is difficult to say whether pleural effusion was due to relapse of previous infection or a re-infection. It is also not known whether TB patients remain susceptible to yet another infection in some other extra-pulmonary site.

Sputum induction (in addition to pleural fluid) for acid-fast bacilli and culture is a recommended procedure in all patients with TB pleurisy. Abdominal TB should be suspected in patients with fever, abdominal pain and ascites. This condition carries good prognosis, if promptly diagnosed and treated [17–19]. A reasonable management strategy for pleural TB would be to initiate a four-drug regimen and perform a therapeutic thoracocentesis in patients with large, symptomatic effusions.

Competing interests

The author declares that there are no competing interests.

Authors' contributions

TH monitored the patient during the illness, treatment and follow-up and has written the manuscript.

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References

[1] D. Dev, G.S. Basran, Pleural effusion: a clinical review, Monaldi Arch. Chest Dis. 49 (1) (1994) 25–35.
[2] D.D. Storey, D.E. Dines, D.T. Coles, Pleural effusion. A diagnostic dilemma, JAMA 236 (19) (1976) 2183–2186.
[3] J.M. Porcel, Tuberculous pleural effusion, Lung 187 (5) (2009) 263–270.
[4] A.F. Seibert, J. Haynes Jr., R. Middleton, J.B. Bass Jr., Tuberculous pleural effusion. Twenty-year experience, Chest 99 (4) (1991) 883–886.
[5] L. Valdés, D. Alvarez, J.M. Valle, A. Pose, José E. San, The etiology of pleural effusions in an area with high incidence of tuberculosis, Chest 109 (1) (1996) 158–162.
[6] M. Salazar-Lezama, H. Quiroz-Rosales, J.L. Bañales-Méndez, M. Sánchez-Guzmán, H. Villarreal-Velarde, R. Báez-Saldaña, E. Azcona-Martínez, M. Selman-Lama, Diagnostic methods of primary tuberculous pleural effusion in a region with high prevalence of tuberculosis. A study in Mexican population, Rev. Invest. Clin. 49 (6) (1997) 453–456.
[7] G.T. Kinasewitz, A.P. Fishman, R.H. Winterbauer, S.A. Sahn, Pleural dynamics and effusions; non-neoplastic pleural effusions; malignant pleural effusions, in: A.P. Fishman (Ed.), Pulmonary Diseases and Disorders, 1988, pp. 2117–2170.
[8] K.L. Eibenberger, W.I. Dock, M.E. Ammann, R. Dorffner, M. F. Hörmann, F. Grabenwöger, Quantification of pleural effusions: sonography versus radiography, Radiology 191 (3) (1994) 681–684.
[9] A. Hirsch, P. Ruffie, M. Nebut, J. Bignon, J. Chrétien, Pleural effusion: laboratory tests in 300 cases, Thorax 34 (1) (1979) 106–112.
[10] S.A. Sahn, The differential diagnosis of pleural effusions, West J. Med. 137 (2) (1982) 99–108.
[11] T. Bartter, R. Santarelli, S.M. Akers, M.R. Pratter, The evaluation of pleural effusion, Chest 106 (4) (1994) 1209–1214.
[12] M.C. Houston, Pleural fluid pH: diagnostic, therapeutic, and prognostic value, Am. J. Surg. 154 (3) (1987) 333–337.
[13] L.T. Yam, Diagnostic significance of lymphocytes in pleural effusions, Ann. Intern. Med. 66 (5) (1967) 972–982.
[14] M. Estenne, J.C. Yernault, A. De Troyer, Mechanism of relief of dyspnea after thoracocentesis in patients with large pleural effusions, Am. J. Med. 74 (5) (1983) 813–819.
[15] D.R. Grogan, R.S. Irwin, R. Channick, V. Raptopoulos, F.J. Curley, T. Bartter, R.W. Corwin, Complications associated with thoracocentesis. A prospective, randomized study comparing three different methods, Arch. Intern. Med. 150 (4) (1990) 873–877.
[16] T. Hussain, Grade-III paraplegia in spinal tuberculosis: follow up of a case report and review of literature, J. Clin. Diagnos. Res. 8 (3) (2014) 148–150.
[17] R. Bataller, P. Ginès, V. Arroyo, Practical recommendations for the treatment of ascites and its complications, Drugs 54 (4) (1997) 571–580.
[18] M. Navasa, J. Rodés, Management of ascites in the patient with portal hypertension with emphasis on spontaneous bacterial peritonitis, Semin. Gastrointest Dis. 8 (4) (1997) 200–209.
[19] B.A. Runyon, Care of patients with ascites, New Eng. J. Med. 330 (1994) 337.